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RISK ANALYSIS OF TCDD CONTAMINATED SOIL

by

John Schaum

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This paper provides a methodology for estimating the human exposure and cancer risk associated with 2,3,7,8-TCDD contaminated soil. Five exposure pathways are addressed: dust inhalation, fish ingestion, dermal absorption, soil ingestion, and beef/dairy products ingestion. For each pathway, factors describing contact rate, absorption fraction, and exposure duration are presented along with the equations for calculating exposure levels and associated cancer risk. The methodology features the use of nomographs to provide quick and approximate estimates of risk. More detailed procedures are also provided for more accurate estimates.

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FOREWORD

The Exposure Assessment Group (EAG) of EPA's Office of Research and Development has three main functions: 1) to conduct exposure assessments; 2) to review assessments and related documents; and 3) to develop guidelines for Agency exposure assessments. The activities under each of these functions are supported by and respond to the needs of the various EPA program offices. In relation to the third function, EAG sponsors projects aimed at developing or refining techniques used in exposure assessments. This study is one of these projects and was done for the Office of Solid Waste and Emergency Response.

Dioxin problems first surfaced in the U.S. in the early 1970's with Agent Orange and the Missouri Horse Arenas. Since then dioxin contamination has been found elsewhere in Missouri, Arkansas, Michigan, New York, and New Jersey. EPA has become increasingly involved in the discovery, assessment and clean-up of these sites. The purpose of this document is to provide an exposure and risk estimation methodology for specific application to dioxin contamination sites. This methodology will help us set priorities and make decisions required to address this important problem.

James W. Falco, Director
Exposure Assessment Group

ABSTRACT

This paper provides a methodology for estimating the human exposure and cancer risk associated with 2,3,7,8-TCDD contaminated soil. Five exposure pathways are addressed: dust inhalation, fish ingestion, dermal absorption, soil ingestion, and beef/dairy products ingestion. For each pathway, factors describing contact rate, absorption fraction, and exposure duration are presented along with the equations for calculating exposure levels and associated cancer risk. The methodology features the use of nomographs to provide quick and approximate estimates of risk. More detailed procedures are also provided for more accurate estimates.

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RISK ANALYSIS OF TCDD CONTAMINATED SOIL*

The purpose of this report is to present a procedure for estimating the human exposure and health risks associated with 2,3,7,8-TCDD (referred to as TCDD in remainder of report) contaminated soil. This report was prepared in response to the mandate under the Dioxin Strategy (EPA, 1983).

1.0 APPROACH

This report provides procedures for estimating the human exposure and cancer risk occurring to people living around a site where the soil is contaminated with TCDD. Five exposure pathways are covered: dust inhalation, fish ingestion, dermal absorption, soil ingestion and beef/dairy products ingestion. A two tiered approach is used in the assessment procedure for each of these pathways. The first tier requires minimal data and uses a nomograph to facilitate the calculations. It provides a quick and very approximate estimation of upper-bound risk. The second tier requires more data and involves more complex calculations, but provides more realistic estimates of risk.

The procedures described in this report involve a number of important limitations/assumptions. Exposure calculations require knowledge of the contaminant level at the point of exposure, i.e. contaminant level in air where it is breathed, or water where it is drunk, etc. Typically these values are either measured directly or estimated using source release rates and fate/transport models. The presentation of these techniques are beyond the scope of this paper. Thus, these procedures assume the user can obtain this information independently.

*An interim version of this report was issued in March 1984. This edition modifies and significantly expands the first report.

Additionally, this paper does not discuss the health effects associated with TCDD nor the derivation of the cancer potency estimate. Instead, the paper emphasizes how to estimate human exposure and merely presents the mechanics of how to estimate cancer risk. References are provided for readers desiring further background on the health effects and cancer potency estimates for dioxin.

1.1 Estimation of Cancer Risk

The general procedure for calculating cancer risk, as used throughout this report, is as follows:

$$\text{Cancer Risk} = 1 - \exp(-\text{potency factor} \times \text{exposure}) \quad (1)$$

The cancer potency factor (or 95% upper-limit of the linear slope factor) for TCDD is .156 $(\text{ng/kg/day})^{-1}$. The derivation of this factor is described in EPA, 1984 and further background on TCDD carcinogenicity is provided in EPA, 1981. Exposure has reciprocal units to the cancer potency factor or ng/kg day in this case.

In order to use the above equation properly, it is important that the potency factor and exposure handle absorption in a consistent fashion. The exposure estimates presented in this report represent the amount of contaminant absorbed into the body. The potency factor, however, was derived on the basis of the administered dose (total fed to animals). Thus, an adjustment is needed to make these terms consistent. The potency factor was derived from a study where the TCDD was administered to rats via their feed. Fries and Marrow (1975) report that 50-60% of TCDD in feed is absorbed into rats. Accordingly, the potency based on administered dose must be multiplied by 1.7-2 to give an absorbed dose potency. This adjustment makes the potency and exposure estimates consistent and is used in all risk calculations in this report.

1.2 Estimation Of Human Exposure

In order to facilitate cancer risk calculations, exposure as used in this report, is expressed as a daily dose rate averaged over an individual's lifetime and bodyweight (typical units are ng/kg-day):

$$\text{Lifetime Average Exposure} = \frac{\text{TCDD Concentration} \times \text{Contact Rate} \times \text{Exposure Duration} \times \text{Absorption Fraction}}{\text{Body Weight} \times \text{70 yr lifetime}} \quad (2)$$

The TCDD concentration refers to the concentration of TCDD in the medium of concern at the point where exposure occurs. The medium of concern varies according to the exposure pathway, in air for dust inhalation, fish for fish ingestion, etc. Although this equation represents the general approach used in this report, some refinements were made. For the Tier 1 calculations, a new term called the conversion factor was introduced. This term is defined as:

$$\text{Conversion Factor} = \frac{\text{TCDD concentration in medium of concern at exposure point}}{\text{TCDD concentration in soil at the original source}} \quad (3)$$

The original source, as used in Equation 3, refers to the original source of TCDD contamination. The product of the TCDD concentration in soil at the original source an the conversion factor were substituted in Equation 2 for the TCDD concentration. This factor represents the reductions in dioxin concentration as it moves away from the source. It was introduced to facilitate the development of nomographs, which relate the TCDD concentration in the soil at the site to the resulting cancer risk levels. The nomographs simplify the mechanics of these calculations and help decision makers analyze the potential risk caused by a site or the level of site cleanup needed to achieve certain risk levels. This approach requires estimation of the conversion factor which can be very difficult. Basically, it involves either environmental monitoring or fate/transport modeling or some combination of these. A detailed discussion of these procedures is

beyond the scope of this report. However, a companion report is available which provides guidance for how to estimate conversion factors (Dawson et al. 1984).

The Tier 2 calculations are based on refinements to Equation 2 which allow modifications to reflect site specific conditions and account for the temporal variability in certain parameters. For example, under some circumstances TCDD in soil degrades, which means the exposure levels diminish over time. Additionally, the behavior patterns of the exposed population may suggest different contact rates or exposure durations than assumed in the Tier 1 approach. In summary, the Tier 2 calculations are based on general equations and make fewer apriori assumptions regarding parameter values. This allows adjustment of any of the parameters to match site-specific conditions. The resulting modifications to Equation 2 make it more complex but allow more accurate and realistic estimations of risk. The Tier 2 equations differ slightly for each exposure pathway. The details of how to apply the Tiers 1 and 2 methods are described in Sections 3-7 which cover each pathway separately.

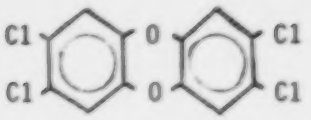
2.0 EXPOSURE ROUTES

TCDD has a very low water solubility, low vapor pressure and strong tendency to sorb on solids (see Table 1). Thus, any transport from the contaminated areas will occur almost entirely in the solid phase.* This report considers only transport by wind blown dust and suspended solids in run-off. Although suspended solids can be carried via the ground water in highly fractured or

*Recent unpublished work by Freeman and Schroy (1984) suggests that volatilization of TCDD in soil occurs rapidly. Since peer review and final publication of this work has not yet occurred, vapor exposure is not addressed in this report.

TABLE 1. PROPERTIES OF TCDD

Structure of 2,3,7,8-TCDD:



Molecular Weight: 322

*Vapor Pressure: 10^{-6} mmHg (estimated)

Solubility in Water: 0.2 ug/l

Octanol-Water Partition Coefficient: 6.9×10^{6} (calculated)

Source: Mabey, et al. 1981

* An unpublished paper by Schroy et. al. (1984) reports the vapor pressure at 25°C as 1.5×10^{-9} mm Hg.

porous strata, such areas are relatively uncommon. Additionally, the presence of a liquid organic phase, which may occur at disposal sites, would enhance the transport of TCDD through soil. Although, such transport appears possible, it is assumed to be relatively uncommon and is not addressed here.

The run-off and dust transport routes could cause human exposure in a number of ways:

- o Direct inhalation of dust.
- o Run-off to streams, accumulation via the aquatic food chain and ingestion of fish. Humans could also contact TCDD while swimming in contaminated waters. This route is considered minor since swimming is generally a relatively infrequent activity and contact with or ingestion of the sediment is minimal.
- o Deposition of dust or eroded soil on residential areas, direct human contact and dermal absorption or ingestion. Although, TCDD is tightly bound to soil, studies have shown that dermal absorption can occur (Poiger and Schlatter, 1980). Dermal contact with soil could result during outdoor recreation or gardening and yard work. Soil ingestion can occur particularly among young children with mouthing tendencies. Home grown vegetables could also become contaminated with TCDD, but this contamination is diminished by several factors. TCDD is generally not taken up significantly in plants* and vegetables are typically washed to remove deposited dust before consumption. Additionally, except in dry and disturbed areas, relatively little dust transport occurs.

*Some investigators (Cocucci et al. 1979) have found evidence of low levels of TCDD plant uptake. However, others (Wipf et al. 1982) could not detect any uptake.

- o Deposition of dust or eroded soil on pastures, accumulation in cattle and ingestion of dairy products or beef. Although this route could also apply to other kinds of livestock, the cattle route is considered most significant since people generally consume more cattle products than other kinds of animal foods and cattle typically graze outdoors where the potential for contact with contaminated soil is greatest.

Based on the above, this paper has focused on dust inhalation, fish ingestion, dermal absorption, soil ingestion, and beef/dairy products ingestion. The exposure scenarios assumed under the Tier 1 calculations for each pathway are summarized below:

- o Dust inhalation, soil ingestion and dermal absorption -- The exposure associated with all three of these pathways is assumed to occur in a residential setting. The soil around the residence, indoor dust deposits, outdoor suspended dust and indoor suspended dust are all assumed to be equally contaminated. Thus the exposure is assumed to occur indoors as well as outdoors. People are assumed to live in this situation for an entire 70 year life. Some adjustments are made for climatic considerations (i.e., frozen soil) which could restrict dust movement or soil contact and associated exposures. Also, soil ingestion is assumed to only occur during ages 2-6 when mouthing tendencies and lack of personal hygiene understanding are highest.
- o Fish ingestion -- For this pathway, it is assumed that a person receives his entire freshwater fish diet from a contaminated source over a 70 year life. This scenario would probably involve a person who lived near a contaminated water body and fished for subsistence purposes.

- o Beef/dairy products ingestion -- Under this pathway it is assumed that a person receives his entire beef and milk diet from a contaminated source over a 70 year life. This situation would probably involve a farmer whose fields were contaminated and derived his beef and milk from livestock raised on his property. Home slaughter is common at many commercial ranches, so this scenario could include more than just subsistence level farmers.

These are obviously worst case assumptions. Many factors such as behavior patterns, climatic conditions, source size, and remedial actions could all reduce potential exposure levels. Such factors can only be considered on a site-specific basis and, therefore, cannot be considered under the generic Tier 1 calculations. However, the Tier 2 calculations are designed to allow considertion of such site-specific conditions and should be used to refine the Tier 1 estimates.

The factors affecting each exposure route are discussed and computational techniques are presented in Sections 3-7. The remainder of this section discusses issues common to all of the exposure routes.

2.1 Body Weight

The exposure calculation for each route requires making a body weight assumption for substitution into Equation 2. The body weight selected should reflect the weight of the exposed individual(s) during the period which they are exposed. The Tier 1 procedures make the apriori assumption that the weight of an average male or 70 kg (Snyder et al. 1975) will generally reflect the actual exposure conditions for all routes except soil ingestion. Since soil ingestion is assumed to be significant during only ages 2-6 the average weight for these

ages of 17 kg (Snyder et al. 1975) was used.

The Tier 2 procedures allow the user to make his own determination of the most appropriate body weight. This decision should be based on the age of the exposed population over the exposure period. Body weight is related to age as follows:

Age (yr)	Body weight (kg)
0-18	3.14 kg + (3.52 kg/yr x age)
>18	70 kg

These relationships describe average male weight and were derived via a regression analysis (Figure 1) on data presented by Snyder et al. (1975). After identifying the ages of exposure, the analyst should integrate the weight over the appropriate ages and divide by the exposure period. This value will best represent the average weight to use in the exposure calculation. For example, if the exposure occurs over a 20 year period when the individual is aged 8-28 the average weight would be 59 kg:

$$\text{Average Weight} = \left[\int_{8}^{18} 3.14 + 3.52 \times dx + (28-18) \quad (70) \right] /20$$

2.2 Lifetime

The exposure calculation for each route also requires making a lifetime assumption for substitution into Equation 2. For compatability with the dose-response estimates dervied from animal studies, this value should represent the total lifetime of the exposed individual. Accordingly, in the Tier 1 calculations it is always assumed equal to 70 yr which represents an average U.S. male. It is also recommended for use in the Tier 2 calculations unless site specific

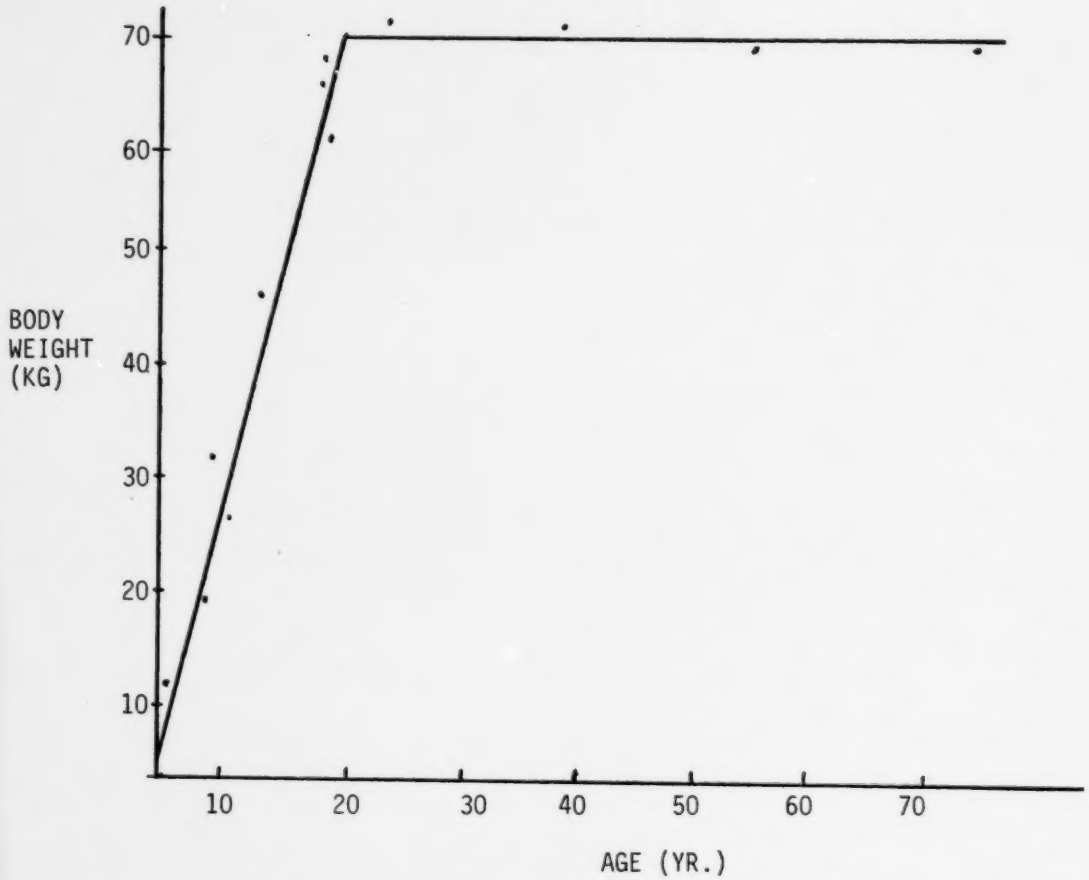


Figure 1. Age vs Body Weight

data on the exposed population suggests a different average lifetime.

2.3 Degradation of TCDD in Soil

The exposure calculations for each route require making a TCDD concentration assumption for substitution into Equation 2. Although this concentration represents the medium of concern at the point of exposure, it is directly dependent on the concentration of TCDD in soil at the original site. If the concentration at the site is changing due to degradation, the concentration at the point of contact will change as well. Accordingly it must be considered when estimating exposure.

The degradation of TCDD in soil is difficult to measure. Most investigators have found that it is generally resistant to biological and chemical degradation, but susceptible to photolytic degradation (EPA, 1984). Young (1983) measured the half-life of TCDD in soil as 10-12 yr and attributes most of the degradation to photodecomposition. This study has adopted this value as a lower limit in the Tier 1 calculations, since it assumes that the TCDD is located at or near the surface and consequently at least partially exposed to sun light.

However, Young states that physical mechanisms such as wind or water erosion could also account for the observed losses. Given this uncertainty and fact that much of the TCDD in soil may not be exposed to sun light, it appears that under some conditions essentially no degradation would occur over the time frame of interest, i.e., 70 yr. Thus, for Tier 1 the half-life is assumed to range from 10 years to infinity. Although, this range appears very wide, actually as the half-life increases over 100 years it has very little impact on the final risk estimate as demonstrated in Figure 2. This figure represents how risk changes when only the half-life is changed, i.e., all other parameters held constant.

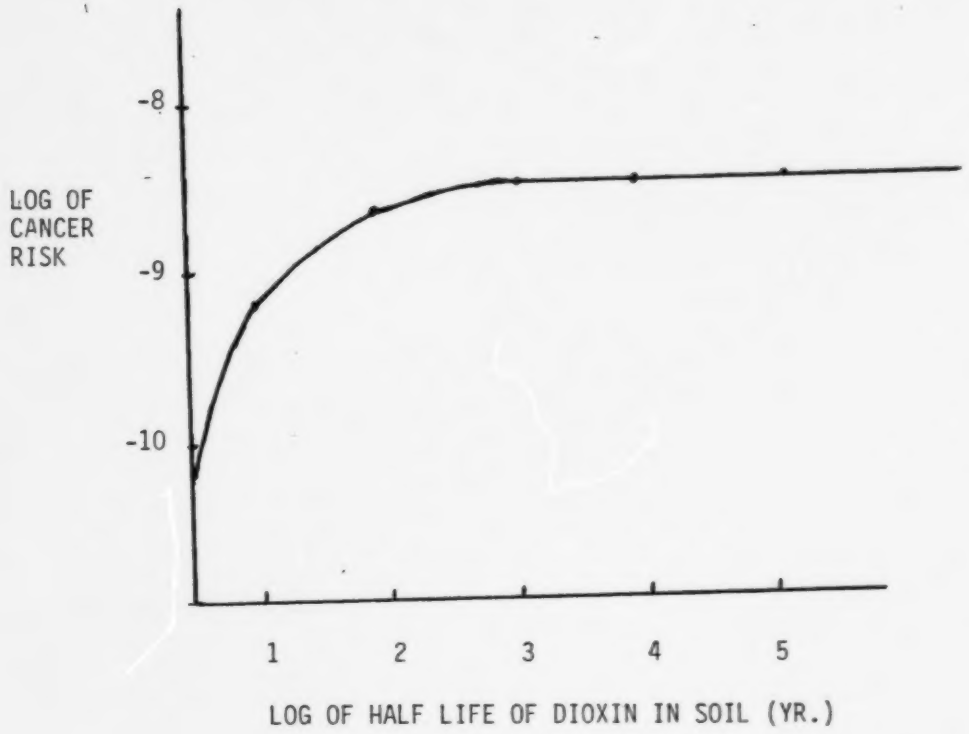


Figure 2. Risk vs Half-Life

Figure 2 also demonstrates that the risk estimate is sensitive to the half-life choices under 100 yr.

Under Tier 2 the analyst should choose a half-life most representative of the site. If monitoring data is available, they may show trends which can be used to estimate degradation rates. Alternatively it is recommended that a half-life near 10 years be chosen if the TCDD contamination is at or near the surface and over 100 years if the TCDD is buried deeply (per the previous discussion). Once a half-life has been selected the exposure is calculated under the assumption that the concentration will vary according to first order kinetics:

$$\frac{dC}{dt} = kC \qquad (4)$$

where, C= concentration
t= time
k= degradation rate constant

$= \log_e .5/T_{1/2}$

$T_{1/2}$= half-life

The concentration at any point in time is calculated by solving Equation 4:

$$C = Co\ e^{-kt} \qquad (5)$$

where, Co = initial concentration

Using Equation (5) the exposure can be calculated by integrating C over the exposure time and substituting into Equation (2). Alternatively, exposure can be estimated by solving for C at frequent intervals, computing exposure, and summing exposure values. Simple calculator programs should be used to conduct such calculations.

The effects of degradation on exposure can be determined as shown below:

$$\frac{\text{Degradation Exposure}}{\text{Non-degradation Exposure}} = \frac{\int Cdt}{Cot} = \frac{(1-e^{-kt})}{kt} \qquad (6)$$

In the Tier 1 calculations the exposure is multiplied by this ratio to reflect the effects of degradation. The ratio will always have an upper limit of 1 when it is assumed that degradation will not occur (i.e., half-life equals infinity). The lower limit is calculated using a 10 yr half life and the upper end of the exposure duration assumption which provides the maximum degradation.

The above discussion assumes that degradation will occur according to first order kinetics. Although, this assumption is commonly applied to these types of problems, it is generally recognized as an over simplification of a very complex problem. Recent unpublished work by Freeman and Schroy (1984) suggests that TCDD degradation in soil follows much more complex kinetics due largely to relatively rapid and strongly temperature dependent volatilization. Since peer review and final publication of the work has not yet occurred, no final conclusions can be drawn. However, it is potentially very important in two respects:

- o It may mean that a first order kinetics approach to this problem is inappropriate.
- o It may mean that potential exposure periods are much shorter than previously thought.

2.4 Exposed Populations

The monitoring data or modeling results will probably show that TCDD concentration in the environment diminishes with distance from the original

contamination source. Similarly, the exposure and risk levels will diminish with distance. The exposure and risk estimates can be plotted on a map and isopleths constructed. These lines show areas with equal exposure or risk levels and can be used to identify how many people are exposed at various levels. In sparsely populated areas, U.S. Geographical Survey maps should be used since they show individual buildings. Local officials should be consulted to determine how many people are associated with such buildings. Otherwise an average of 3.8 persons/dwelling should be assumed. In more dense areas, the best population statistics are available from the Bureau of Census. Population estimates for counties and smaller areas are provided by the Bureau at (202) 763-5002.

2.5 Risk from Combined Exposure Routes

The procedures described in this study explain how to calculate the risk associated with individual exposure routes. In situations where an individual is exposed to TCDD by more than one pathway, the risks should be calculated separately and then added.

3.0 DUST INHALATION

Dust is generated from land surfaces as a result of mechanical disturbances (i.e., vehicle traffic) or wind erosion and dispersed via the wind. Cowherd et al. (1984) have recently completed a manual specifically for the purpose of estimating dust emission rates from contaminated land surfaces and resulting air concentrations around the source. If sufficient monitoring data is unavailable, it is highly recommended that analysts consult this manual to model dust emissions.

TCDD, as discussed earlier, is typically tightly bound to soil particles.

The organic carbon content and surface area of the particles affect how much TCDD absorbs to particles. Since these factors may vary between the source soils and dust generated from them, the TCDD levels may also differ. Unfortunately, the influence of these factors are generally not known and it is typically assumed that the TCDD levels in the soil and dust are equal:

$$\begin{matrix}\text{TCDD level} \\ \text{in air}\end{matrix} = \begin{matrix}\text{TCDD level} \\ \text{in soil}\end{matrix} \times \begin{matrix}\text{Total particutate} \\ \text{Concentration in air}\end{matrix} \qquad (7)$$

It is recommended that this assumption be used in determining the concentration values substituted into Equation 2 under the Tier 2 calculation or into the nomograph (Figure A-1) when making Tier 1 calculations. The conversion factor for the Tier 1 calculation is defined as follows:

$$\text{Conversion Factor} = \frac{\text{TCDD concentration in air at exposure point } (ng/m^3)}{\text{TCDD concentration in soil at original source } (ng/g)} \qquad (8)$$

3.1 Contact Rate

The contact rate for this exposure route is an individual's respiration rate. Snyder (1975) estimates the inhalation rate for an average adult male as 7.5 l/min while resting and 20 l/min during light activity. Snyder further estimates that an average adult spends 8 hr/day resting and 16 hr/day engaged in light activity. Thus the average daily inhalation rate is:

$$\begin{matrix}\text{Daily} \\ \text{Inhalation} \\ \text{Rate}\end{matrix} = \frac{60 \text{ min/hr } [(8 \text{ hr/day} \times 7.5 \text{ l/min}) + (16 \text{ hr/day} \times 20 \text{ l/min})]}{1000 \text{ l/m}^3}$$

$$= 23 \text{ m}^3\text{/day}$$

This is the value used for the Tier 1 calculations which conservatively assumes that individuals will be exposed 24 hr/day. Under Tier 2 calculations, the analyst should adjust this value if the site specific conditions suggest that exposure occurs during only a portion of the day.

3.2 Exposure Duration

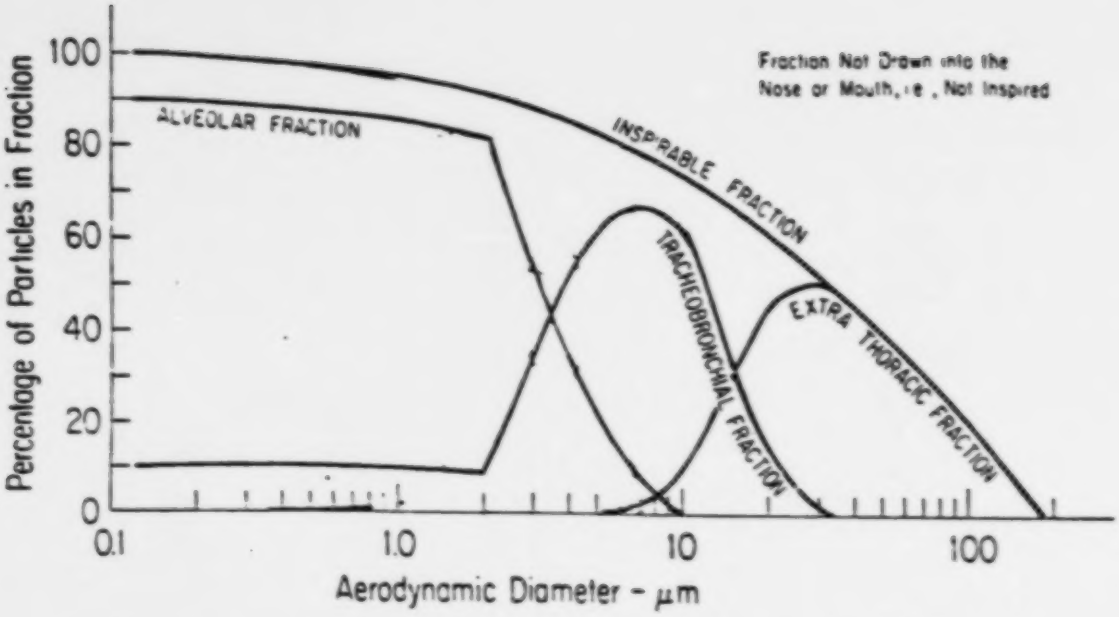
Dust generation and the resulting exposure is essentially eliminated when the soil is very wet or frozen. Obviously, such conditions will vary widely across the country. In warm arid areas such as the southwest, the conditions preventing dust emissions almost never occur. Whereas, in Minneapolis, the soil is frozen an average of 118 days/yr (Personal communication from Don Baker, Minnesota State Climatoligist, April 2, 1984). Cowherd, et al. (1984) suggests that dust emissions are negligible on days when precipitation exceeds .01 inches which is reported by Cowherd as 110 days/yr for Minneapolis. NOAA (1980) suggests that approximately 80% of the precipitation days occur outside of the winter months. Thus, Minneapolis has a total of 206 days/yr (118 + 80% of 110) when the soil conditions would prevent dust emissions. For purposes of the Tier 1 calculations it was assumed that the arid Southwest and Minneapolis would represent the possible range of conditions. Accordingly, under Tier 1 the exposure duration was assumed to vary from 159 to 365 days/yr or a total of 11,130-25,550 days over a 70 yr life.

Under Tier 2, the user should adjust the exposure duration to reflect the climatic conditions of the site. Such adjustments should only be made if they are not accounted for elsewhere. The models presented by Cowherd et. al. (1984) adjust the emission rate estimates on the basis of climatic conditions. If such models are used it would be redundant to also adjust the exposure duration on the same basis. The behavior patterns of the exposed population can also effect the exposure duration and should be adjusted accordingly, if the appropriate data is available. Finally, a mass balance should be conducted to ensure that mass of contaminant emitted does not exceed the amount present.

3.3 Absorption Fraction

For this route the absorption fraction is the fraction of the contaminant entering the lungs which is absorbed into the body. The fraction of particles which are inspired (i.e. enter the respiratory system) depend on numerous factors such as breathing rate, particle size distribution, wind speed and whether breathing is done through the mouth or nose. The International Standards Organization (1981) has estimated the inspired fraction as a function of particle size under average conditions (Figure 3). Particle sizes <10 u are generally considered most important in estimating health effects. Virtually all of these particles will be inspired. However, their fate after entering the lungs is less certain. Generally, the heavier particles deposit in the upper regions of the respiratory tract, the lighter particles in the lower regions and the very lightest are exhaled. Most of the deposited particles in the upper regions and some in the lower region are clearred by ciliary action and swallowed. Lacking specific particle size distribution information the fate of inspired particles should be assumed to follow the recommendations of the International Commission on Radiological Protection (Table 2). Since TCDD has a low water solubility, the recommendations for "other compounds" would apply.

After determining how much of the particles are swallowed, the overall absorption fraction can be further refined on the basis of GI tract absorption. Poiger and Schlatter (1980) found that 13.8 - 18.2% of the orally administered TCDD (which had been absorbed to soil for 8 days) reached the liver in 24 hr. Assuming that this represents 70% of the body burden (Fries and Marrow, 1975) the total GI tract absorption is 20-26%. McConnell, et al. (1984) also found that the absorption of TCDD from soil in the GI tract was "highly efficient"



The Alveolar Fraction represents the particles reaching the alveoli (ie. deepest region of lungs). The Tracheobronchial Fraction represents the particles reaching the tracheobronchial system (ie. central region of lungs). The Extra Thoracic Fraction represents the particles reaching the area outside the thorax (ie. nose and throat).

Figure 3. Inspired Fraction vs. Particle Size

Source: International Standards Organization, 1981.

Table 2. Distribution of Inspired Particles

	Readily soluble compounds (%)	Other compounds (%)
Exhaled	25	25
Deposited in upper respiratory passages and subsequently swallowed	50	50
Deposited in the lungs (lower respiratory passages)	25 (this is taken up into the body)	25*

* Of this, half is eliminated from the lungs and swallowed in the first 24 hr, making a total of 62.5% swallowed. The remaining 12.5% is retained in the lungs with a half-life of 120 days, it being assumed that this portion is taken up into the body fluids.

Source: International Commission on Radiological Protection, 1968.

in test animals suggesting that at least this much is absorbed.

In summary, the overall absorption fraction is calculated as follows:

$$\text{Absorption Fraction} = \text{Inspired Fraction} \left[\text{Fraction Remaining in Lungs} + \left(\text{Fraction Swallowed} \times \text{GI Tract Absorption Fraction} \right) \right] \quad (9)$$

Using Equation 9, an absorption fraction of .25-.29 was derived from the following assumptions:

Inspired Fraction = 1.0 (on basis that <10 u particles are primary concern)

Fraction Remaining in Lungs = .125 (ICRP, 1968)

Fraction Swallowed = .625 (ICRP, 1968)

GI Tract Absorption = .20-.26 (Poiger and Schlatter, 1980)

This value is also recommended for Tier 2 calculations unless site-specific data suggests otherwise.

4.0 FISH INGESTION

Fish contamination results from the transport of eroded soil via runoff from the TCDD contaminated site to local surface waters. The contaminated soil mixes with the other sediment in the water body lowering the effective TCDD concentration. In a river or stream the TCDD moves downstream with the sediment and the concentration of TCDD decreases further due to dilution with clean sediment. This process is very complex and highly site specific. Once TCDD has entered a water body, it has been shown to bioconcentrate in aquatic species.

The nomograph for the Tier 1 calculations is presented in Figure A-2. The conversion factor used for this exposure route is defined as:

$$\text{Conversion Factor} = \frac{\text{TCDD concentration in Sediment Where Fish are Caught (ng/g)}}{\text{TCDD Concentration in Soil at Original Source (ng/g)}} \quad (10)$$

4.1 Bioaccumulation

The approach taken in this study assumes that equilibrium conditions have been reached and remain constant over the exposure period. This means that the TCDD levels in the sediment remain constant at a particular point and that the fish have reached an equilibrium with the environment. This further implies that the levels of TCDD in fish from a certain area will remain constant over time and at a constant relationship to the TCDD level in the sediment. In reality, some species such as bottom feeders will move toward equilibrium conditions faster than others, many species may never reach equilibrium due to the fact that they do not spend enough time in one location, and finally some species will bioconcentrate more TCDD than others due to greater lipid content. For Tier 1 the ratio of TCDD in fish to TCDD in sediment is assumed to range from 1 to 10 as reported by Kenaga and Norris (1983).

For Tier 2, the analyst should attempt to find data on the fish species and water body conditions that best reflect the site being analyzed.

4.2 Consumption Rate

For the Tier 1 calculations the consumption rate is assumed equal to 6.5 g/day which is the U.S. averge for fresh water fish (Stephan, 1980). The average may be higher in areas near large fish supplies, such as the Great Lakes region. Additionally, the average consumption rate among fish eaters will be higher than the overall average. However, we generally lack the necessary data to reflect these phenomena. Unless site-specific data is available it is recommended that 6.5 g/day be used in the Tier 2 calculations as well.

4.3 Exposure Duration

For this pathway the exposure duration represents the number of days that the exposed population eats contaminated fish. For the Tier 1 calculations it is conservatively assumed that this will occur every day of an entire 70 year life or 25,550 days. In reality this value is probably much less due to several factors:

- o Few individuals receive their entire fish diet from fish caught in one location.
- o Seasonal conditions may prevent catching fish from the contaminated area.
- o The contamination source may not last 70 years. A mass balance should be conducted to determine how long it could last.

The analyst should attempt to consider these factors in selecting an exposure duration for use in the Tier 2 calculations.

4.4 Absorption Fraction

For the Tier 1 calculations the fraction of TCDD in fish which is absorbed in the GI tract was assumed to range from .5 to .86. This was based on the following two studies as reported by McConnell et al. (1984):

- o 50-60% of TCDD in diet of rats was absorbed.
- o 86% of TCDD in a mixture of acetone and corn oil fed by gavage to rats was absorbed.

Use of these data assumes that absorption from TCDD in fish will be similar to absorption from TCDD in rat food and acetone/corn oil mixture.

This range is recommended for use in Tier 2 calculations as well unless more relevant data becomes available.

5.0 DERMAL ABSORPTION

Deposition of contaminated dust or eroded soil in residential areas can cause human exposure by direct contact and dermal absorption.

The conversion factor used in the Tier 1 calculations for this route is defined as:

$$\text{Conversion factor} = \frac{\text{TCDD concentration in soil at exposure point (ng/g)}}{\text{TCDD concentration in soil at original source (ng/g)}} \quad (11)$$

The nomograph is presented in Figure A-3.

5.1 Contact Rate

The amount of soil which accumulates on people's skin will depend on numerous factors such as behavior characteristics, type of soil, soil conditions (i.e., moisture, temperature, etc.) exposed skin area, contact time, etc. The variability in these factors and lack of data make estimating dermal soil accumulation very difficult. The approach below is based on minimal data and does not account for all of the above factors. Thus, although it represents our current best estimate, much uncertainty remains.

The amount of soil which accumulates on skin was estimated from studies by Lepow (1975) and Roels (1980).

Lepow (1975) found that children accumulated at least 11 mg of soil on their hands after normal playing in and around their residences. This estimate was based on soil samples collected by pressing a 21.5 cm^2 tape against the hands of the children. Obviously, this method is not 100% efficient and Lepow indicated that the samples collected represented only a small fraction of the total soil on their hands. For purposes of this analysis it was assumed that this measurement represented a lower bound estimate for the amount of soil on a

21.5 cm^2 area. Thus, the average soil level was 11 mg/21.4 cm^2 or 0.5 mg/cm^2. Roels et al. (1980) measured the amounts of Pb on the hands of children by rinsing the palm and fingers of one hand with dilute nitric acid. The levels of Pb in the soil and dust where the children lived are also measured. Using these data the amount of soil on the hands was calculated as follows:

$$\text{Amount of soil per unit area} = \frac{\text{Amount of Pb/hand}}{(\text{Concentration of Pb in Soil})\ (\text{Surface Area of Hand})} \quad (12)$$

the surface area of the hand was determined by Snyder (1975) who suggests that the palm and fingers of one hand comprise 1% of the total body surface area. Snyder (1975) also gives the total surface area of 11 year old children (average age studied by Roels) as 10165 cm^2. Thus, the area of the palm and fingers was assumed equal to 102 cm^2. Substituting this value and the Roel's data into the above formula the amount of soil on the skin was calculated to be 1.5 mg/cm^2. This level was assumed to represent an upper estimate producing an overall daily contact range of 0.5 to 1.5 mg/cm^2. Additionally, it was assumed that this range represents an average value for the entire exposed area of the body. Normally hands are probably dirtier than other parts of the body, but the fact that neither of the hand measurement techniques are 100% efficient makes it a more reasonable estimate for the average value of the entire exposed area. It was further assumed that this range applies to adults working outdoors as well as children. Unless other data are available, it is recommended that this range be used for Tier 2 as well as Tier 1 calculations. Since this contact rate is expressed in per unit area terms, it must be used in conjunction with estimates of the exposed surface area, which is discussed in Section 5.3.

5.2 Exposure Duration

For this route, the exposure duration represents the number of days that an individual will contact the contaminated soil. In a residential setting behavior patterns and seasonal conditions will most influence this parameter. Children who enjoy playing outdoors and adults who enjoy gardening or other types of yard work could contact soil very frequently. In warm climates such people could contact soil every day. In the coldest parts of the U.S. such as Minneapolis, the soil is frozen an average of 118 days/yr (Personal Communication from Don Baker, Minnesota State Climatologist, April 2, 1984). Although, other types of inclement weather, illness, travel and other factors could reduce the duration period, no data could be found clearly connecting these phenomena to the potential for soil contact. Accordingly, the range of 247-365 days/yr was adopted for the Tier 1 calculations. Under Tier 2, the analyst should attempt to find site specific data for refining this number. The exposure duration can also be affected by the source size which should be analyzed via a mass balance.

5.3 Exposed Surface Area

The exposed surface area of an adult has been estimated by Sendroy (1954) as:

- o 2940 cm^2 - wearing short-sleeved, open-necked shirts, pants, shoes, with no gloves or hats.
- o 910 cm^2 - wearing long-sleeved shirts, gloves, pants and shoes.

The exposed surface area of children was computed by multiplying the adult values by the ratio of a child's total surface area to an adult's total surface

area.

Based on the above assumptions, the total amount of soil which accumulates on the exposed area of people was computed as follows:

Total Accumulated Soil = (Contact rate)(exposed surface area)(exposure duration)

Since the surface area changes with age, this calculation has to be made for each year and summed over a lifetime. As shown in Table 3, this approach yields an estimate of the potential lifetime soil accumulation of 7,900 - 110,000 g which was adopted for the Tier 1 calculations. The exposure duration assumption will probably make this estimate unrealistically high in most situations. Therefore it is strongly recommended that users attempt to find site specific data to refine this estimate under the Tier 2 calculations.

5.4 Absorption

Poiger and Schlatter (1980) found that 0.05 to 2.2% of the TCDD in a soil paste applied dermally to laboratory animals for 24 hr reached the liver. Poiger and Schlatter also reported that other investigators found about 70% of the total TCDD body burden in the liver. This suggests that the total absorption actually varied from 0.07 - 3%. This range was selected for use in the Tier 1 calculations. Unless other data is available it is recommended for use in the Tier 2 calculations as well. Such extrapolations from animals to humans introduces uncertainty due to differences in skin properties.

6.0 SOIL INGESTION

Deposition of contaminated dust or eroded soil in residential areas can also cause human exosure by ingestion. Although soil ingestion occurs throughout

TABLE 3. AMOUNT OF SOIL ACCUMULATED DURING LIFETIME

Age	Exposure Duration[1] (days)	Total Surface Area[2] (cm^2)	Surface Area Child/Adult	Exposed Surface Area[3] (cm^2)	Total Accumulated Soil (g)
2-3	494-730	5800	0.33	300 - 980	74 - 1100
4-5	494-730	7200	0.41	380 - 1200	94 - 1300
6-7	494-730	8100	0.47	420 - 1400	100 - 1500
8-9	494-730	8900	0.51	470 - 1500	120 - 1600
9-10	494-730	9300	0.53	490 - 1600	120 - 1800
11-12	494-730	10300	0.59	540 - 1700	130 - 1900
12-13	494-730	11100	0.64	580 - 1900	140 - 2100
13-14	494-730	11700	0.67	610 - 2000	150 - 2200
15-16	494-730	13800	0.79	720 - 2300	180 - 2500
16-17	494-730	14800	0.85	770 - 2500	190 - 2700
17-18	494-730	15500	0.89	810 - 2600	200 - 2800
18-19	494-730	16100	0.93	840 - 2700	210 - 3000
19-20	494-730	16600	0.95	870 - 2800	210 - 3100
20-22	741-1095	17000	0.98	890 - 2900	330 - 4800
22-24	741-1095	17400	1.0	910 - 2900	340 - 4800
24-70	11609-17155	17400	1.0	910 - 2900	5300 - 75000
				Total	7900 - 110,000

1. Exposure Duration = 247-365 day/yr x years of exposure.
2. Snyder, 1975.
3. Lower Estimate

a person's life, it will be most significant during childhood. For this reason and lack of data on how much soil ingestion occurs among adults, this study only estimates exposure to children.

The conversion factor used in the Tier 1 calculations is identical to that used for the dermal absorption route:

$$\text{Conversion Factor} = \frac{\text{TCDD concentration in soil at exposure point (ng/g)}}{\text{TCDD concentration in soil at original source (ng/g)}} \quad (13)$$

The nomograph is presented in Figure A-4.

6.1 Contact Rate

The amount of TCDD-contaminated soil which children may ingest as a result of normal playing around their home is very difficult to estimate. The ingestion rates will depend on the mouthing and pica tendencies of the children.

Based on measurements of the amount of soil found on children's hands and observations of mouthing frequencies, Lepow (1975) estimated that children could ingest at least 100 mg of soil per day. This estimate does not account for direct ingestion of soil which could increase daily ingestion rates to 5 g/day (personal communication from Julian Chisolm, Baltimore City Hospital, November 1982). This range was selected for use in the Tier 1 calculations.

Unless site specific data is available, it is recommended that this range be applied in Tier 2 calculations as well.

6.2 Exposure Duration

For this pathway, the exposure duration represents the number of days that a child consumes contaminated soil. Obviously this number can vary tremendously depending on individual behavior patterns, access to contaminated areas, soil

conditions, etc.

The children studied by Lepow ranged from 2-6 yr old. Lacking other data, it was assumed for purpose of the Tier 1 calculations that this represents the ages that mouthing tendencies and lack of understanding of personal hygiene will cause the most significant soil ingestion. As with the dermal absorption pathway, it was assumed that the soil could be unfrozen from 247-365 days/yr depending on location. Although other types of inclement weather, illness, travel and other factors could reduce and potential duration period, no data could be found reflecting such phenomena. Accordingly for the Tier 1 calculations, the exposure duration was assumed to last 247-365 days/yr from ages 2-6 for a total of 1240-1830 days.

This assumption probably represents a severe worst-case for most situations. For the Tier 2 calculations it is strongly recommended that the analyst attempt to find site specific data leading to more realistic estimates. A mass balance should be conducted to confirm that the source emissions can last at least 5 years.

6.3 Absorption

The GI tract absorption of TCDD in soil has already been discussed under Section 3.3. In summary, an absorption fraction of .20 - .26 (Poiger and Schlatter, 1980) was used for the Tier 1 calculations and is also recommended for the Tier 2 calculations.

7.0 BEEF/DAIRY PRODUCTS INGESTION

The deposition of contaminated dust or eroded soil on pasturelands can lead to uptake in the human food chain and eventual human exposure. The consumption of soil by cattle has been measured to average .72 kg/day (Fries, 1982). Thus, if the soil is contaminated, beef and milk can also become

contaminated. This process can occur relatively quickly. Fries (1982) reports that PCB levels in milk reached steady state three weeks after it was introduced into the diet.

The conversion factor for the Tier 1 calculations is defined as:

$$\text{Conversion Factor} = \frac{\text{TCDD Concentration in Pasture Soil (ng/g)}}{\text{TCDD Concentration in Soil at Original Source (ng/g)}} \quad (14)$$

The nomograph used to facilitate the Tier 1 calculations is illustrated in Figure A-5.

As explained below, the assumptions made for this exposure pathway reflect a situation where a person would obtain his entire beef and milk diet from livestock raised on his property. This is obviously a worst case situation and users should understand that commercial marketing practices would reduce such exposures for most people (discussed below).

7.1 Bioaccumulation

A number of studies have been conducted on chemicals similiar to TCDD such as PCB, PBB, and DDT, which relate the level of the contaminant in the diet to the resulting level in body fat or milk fat. Fries (1982) reports that these compounds reach an upper estimate, steady state fat/diet ratio of approximately 5. Jensen et al. (1981) conducted similar studies using 2,3,7,8-TCDD and found the steady state fat/diet ratio to be approximately 4, which suggests that TCDD behaves similarly to PCB, DDT, and PBB. Using a fat/diet ratio of 5 and data regarding the soil content of the diet, Fries estimates the milk fat/soil ratio as .7 and the tissue/soil ratio as .23. These estimates are based on data from New Zealand where animals are typically grazed throughout the year.

In the U.S. grazing is normally less frequent and supplemental feeds are commonly used. Such feeding practices could alter the amount of soil consumption. Fries and Jacobs (1983) conducted another study where cattle were kept in a feed lot situation containing PBB contaminated soil. Under these conditions, the beef fat/soil ratio averaged .39 and milk fat/soil ratio averaged .40. Since these conditions more typically represent conditions in the U.S., they were selected for use in this study. However, the extrapolation of these results to this assessment involves several important assumptions:

- o TCDD will be metabolized in a similiar fashion to PBB.
- o The portion of soil in the diet is the same in the exposure scenario as the study.
- o Cattle will ingest more TCDD from soil on the ground than from foliage deposits. This is the situation occurring during the experiment from which Fries and Jacobs (1983) derived the .4 fat/soil ratio. Where run-off represents the dominant transport route, most of the TCDD will be on the ground rather than on foliage and this assumption should be valid. However, in dry and disturbed areas, significant dust transport may occur causing cattle to obtain more TCDD from foliage deposits than ground deposits (personal communication from Curtis Travis, Oak Ridge National Laboratory, Feb. 2, 1984). Since, the bioavailability of TCDD in soil may differ from that in foliage deposits, the fat/soil ratio may differ.

In summary, a fat to soil ratio of .4 was adopted for use in the Tier 1

calculations and is also recommended for use in the Tier 2 calculations.

7.2 Consumption Rates

Average beef and milk fat consumption rates and fat content data are presented in Table 4.

This data suggests an average of 62 g/person-day beef and milk fat are consumed. This value was adopted in the Tier 1 calculations and is recommended for Tier 2 as well, unless site specific data suggest otherwise.

7.3 Exposure Duration

The exposure duration for this route refers to the number of days an individual will consume contaminated beef or dairy products. This value can vary tremendously depending on how the contaminated food is distributed. Some individuals may derive all of the beef and milk from the same source which means their exposure duration could potentially last every day of a lifetime. However, most people obtain beef and milk commercially. The production and marketing practices of commercial food operations can greatly reduce exposure durations. For example, milk from a number of dairies may be collected in one truck. Assuming only one dairy is contaminated, the contaminant level is diluted as a result of mixing with uncontaminated milk. Further mixing and dilution may occur at the processing and bottling plant. The milk from one plant probably represents a small portion of the total local market. Thus, an individual is unlikely to buy only milk which was contaminated. Accordingly, that individual's exposure is much less than that suggested by his total milk consumption. Thus, the "dilution" effects of production and marketing reduce individual exposure

TABLE 4. BEEF/DAIRY PRODUCTS INGESTION RATES*

	Total Consumption Rate (g/person-day)	Percentage Fat	Fat Consumption Rate (g/person-day)
Beef	124	15	19
Dairy Products	550	7.8	43
Total			62

*EPA, 1981

levels. This reduction is best estimated using local data. However, the potential dilution is illustrated by following data for the Mid Atlantic Region (Personal Communication from John Buche, Statistical Reporting Service, U.S. Department of Agriculture, Beltsville, MD, November 7, 1984):

One month's production of Class I milk	2.2×10^8 lb
One month's average production per producer	3.2×10^4 lb

These figures suggest a potential "dilution" in exposure level of 6,900 times. Obviously, these numbers represent averages over a large area and different statistics may apply to individual markets. Further study of the dairy and other food industries is needed to more accurately predict the dilution effects caused by production and marketing practices.

These dilution effects can be accounted for by either lowering the assumed contamination levels in food products or reducing the effective duration of exposure to fully contaminated products. For milk it is probably more logical to reduce the contamination levels and assume milk is consumed every day. For beef, it would be better to reduce the duration estimate since the marketing practices will reduce the number of days contaminated beef is eaten rather than the level in the beef.

Unfortunately, we lack the data to characterize these effects and have ignored them in the Tier 1 calculations where it was assumed that the exposure could occur every day of a 70 yr life or 25,550 days. However, the analyst could attempt to consider dilution effects in the Tier 2 calculations, since they appear to have potentially very significant impacts on exposure levels. Other site specific factors such as the source size and accessability could also reduce the exposure duration.

7.4 Absorption

As discussed in Section 4.4, the GI tract absorption used in Tier 1 was assumed to vary from .5 to .86 on basis of studies reported by McConnel et al. (1984). Some uncertainty is introduced by the fact that these studies used rat feed and a mixture of corn oil and acetone rather than beef and milk. Unless better data become available later, it is recommended that this range be assumed for the Tier 2 calculations as well.

8.0 DISCUSSION OF UNCERTAINTY

Users of the methodology described in this report should understand that it involves considerable uncertainty. The uncertainty is derived from the numerous assumptions which may not accurately reflect actual conditions:

- The assumptions regarding body weight, lifetime, and contact rates were based on national averages and may not be representative for specific individuals.
- The absorption data were derived from animal studies and assumed applicable to humans, such extrapolations introduce uncertainty due to differences in the exchange membrane (skin, GI tract, aveoli) properties between animals and humans and differences between the human exposure scenario and experimental design. Such absorption fraction estimates also assume steady-state conditions which may not be achieved in the actual human exposure scenario.
- The exposure duration parameters are based on assumptions regarding behavior patterns and various physical phenomena. These factors are very difficult to estimate, especially in a general rather than site-

specific basis.

The parameter values were typically selected from wide ranges. These ranges were carried through the Tier 1 calculations so that calculated risks show an even greater range of uncertainty. Thus, the final risk estimates reflect the uncertainty associated with the direct parameter assumptions. The magnitude of this uncertainty is expressed by the difference between the low and high estimates of risk given in the nomographs. These differences are summarized below for each exposure pathway:

Exposure Pathway	Orders of Magnitude Uncertainty
Inhalation Exposure	1
Fish Ingestion Exposure	2
Dermal Exposure	2.5
Soil Ingestion	2
Beef/Dairy Products Exposure	1

In addition to the direct assumptions associated with the various parameter values, it is implicitly assumed under Tier 1 that all site conditions which could influence conversion factors (terrain features, climatic conditions, etc.) remain constant over the exposure period.

The uncertainty associated with the Tier 2 calculations should be much less than the Tier 1 calculations since Tier 2 involves fewer apriori assumptions regarding site conditions.

9.0 HOW TO USE NOMOGRAPHS

Nomographs have been developed to faciliate the Tier 1 calculations. One

for each of the exposure routes is provided in Appendix A:

Title	Figure Number
Dust Inhalation	A-1
Fish Ingestion	A-2
Dermal Exposure to Soil	A-3
Soil Ingestion	A-4
Beef/Dairy Products Fat Ingestion	A-5

Each nomograph consists of 3 axes: cancer risk, soil concentration (TCDD level in soil at original source) and conversion factor. The intersection points of a straight line drawn through the axes provides the solution to the problem. Two of the three quantities must be determined before solving for the third. Typically, the conversion factor and soil concentration are known and a risk estimate is desired. This would involve plotting the conversion factor and soil concentration and drawing the line through the points to the risk axis. The intersection point on the risk axis is the risk corresponding to the predetermined soil concentration and conversion factor.

The nomographs were developed by combining Equations 1 and 2 into one equation for risk and making assumptions for all parameter values except the risk, conversion factor and soil concentration. The overall equation, parameter assumptions and conversion factor definition are listed on each nomograph. The parameter assumptions are also summarized in Table 5.

Since some of the parameter values span a range, a range of risk values can be calculated as well. Thus, the risk axis has two scales. The upper risk estimate was derived from parameter values chosen from the ranges to maximize

TABLE 5. SUMMARY OF EXPOSURE FACTORS USED IN TIER 1 CALCULATIONS

	Contact Rate	Absorption Fraction	Exposure Duration	Body Weight	Degradation Effects Ratio	Miscellaneous Factors
Dust Inhalation	23 m^3/day	.25-.29	11,130-25,550 days	70kg	.2-1	
Fish Ingestion	6.5 g/day	.5-.86	25,550 days	70kg	.2-1	Fish-Sediment Distribution Factor = 1-10
Dermal Exposure to Soil	$\frac{7900\text{-}110{,}000\ g}{life}$	.0007-.03	17,290-25,550 days	70kg	.2-1	
Soil Ingestion	.1-5 g/day	.2-.26	1240-1830 days	17kg	.84-1	
Beef/Dairy Products Fat Ingestion	62 g/day	.5-.86	25,550 days	70kg	.2-1	Animal Fat to Soil Biocon-centration Factor = .4

the risk estimate. Conversely, the lower risk estimate was derived from parameter values chosen from the ranges to minimize the risk estimate. Although, the resulting range does not represent all possible uncertainty, it does reflect it to some extent.

In situations where the TCDD concentration is known in the medium of conern (ie. level in air at point inhaled, level in sediment at point where fish caught, etc.), use a conversion factor equal to 1 and the soil concentration (middle axis) equal to the known TCDD concentration in the medium of concern. Depending on the exposure pathway, the middle axis may represent something other than soil concentration. In order to clarify what the middle axis would represent when using this approach the following chart is provided.

Pathway	Middle Axis Representation
Dust Inhalation	Concentration of TCDD in air at exposure point (ng/m^3)
Fish Ingestion	Concentration of TCDD in sediment where fish are caught (ng/g)
Dermal Exposure	Concentration of TCDD in soil at exposure point (ng/g)
Soil Ingestion	Concentration of TCDD in soil at exposure point (ng/g)
Beef/Dairy Products Ingestion	Concentration of TCDD in pasture where animals graze (ng/g)

Finally, it should be noted that the risk equation given on each nomograph is presented in the linear rather than the exponential form as given in Equation 1. These two are equivalent for low exposure values since:

$$1-e^{-qd} = qd \quad , \quad \text{when } qd < 10^{-3}$$

where, q = cancer potency factor

d = dose or exposure

Thus, the nomographs give mathematically correct results only when the risk is less than about 10^{-3}.

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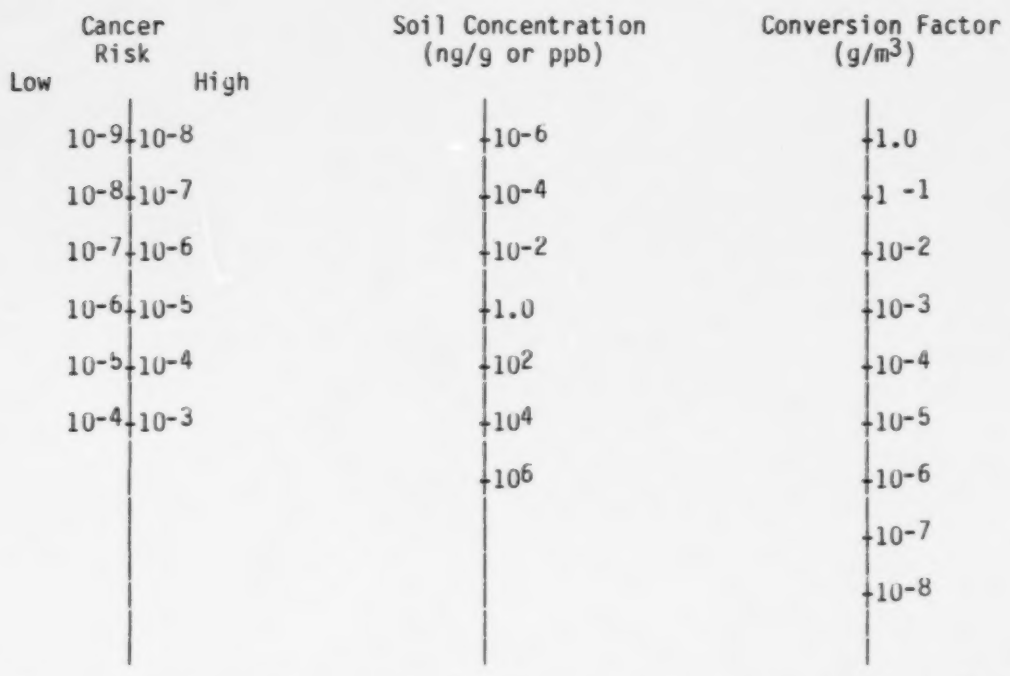
Appendix - Nomographs

FIGURE A-1. NOMOGRAPH FOR INHALATION EXPOSURE

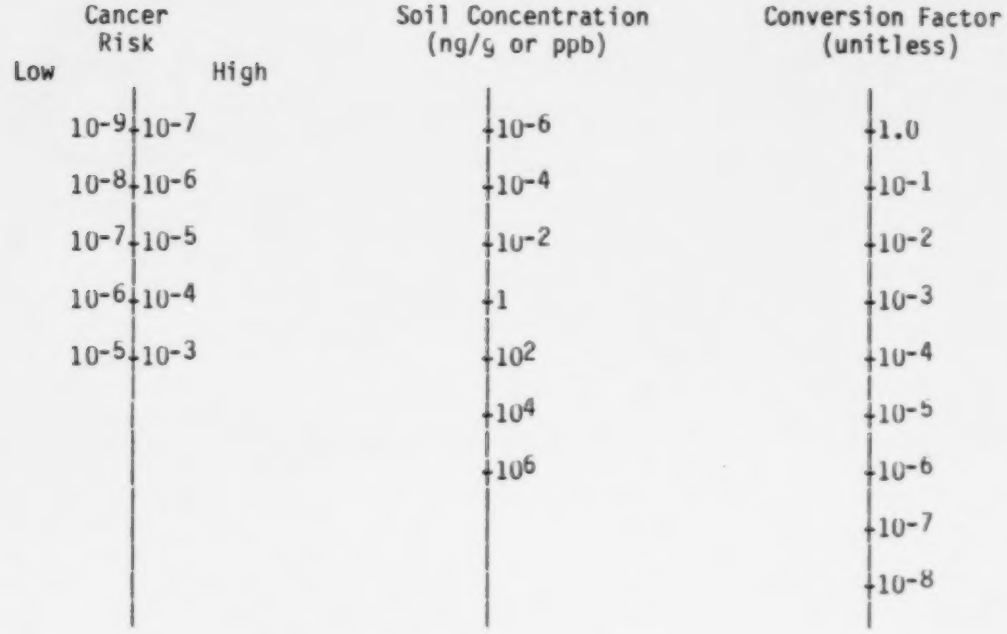


Cancer Risk = (Potency Factor x Soil Concentration x Conversion Factor x Exposure Duration x Inhalation Rate x Absorbed Fraction x Degradation Effects Ratio)/(Body Weight x Lifetime)

where, Potency Factor = .26-.31 $(ng/kg\text{-}day)^{-1}$
Exposure Duration = 11,130-25,550 days
Inhalation Rate = 23 m^3/day
Absorbed Fraction = .25-.29
Degradation Effects Ratio = .2-1
Body Weight = 70 kg
Lifetime = 70 yr

$$\text{Conversion Factor} = \frac{\text{Concentration of TCDD in Air at Exposure Point } (ng/m^3)}{\text{Concentration of TCDD in Soil at Original Source } (ng/g)}$$

FIGURE A-2. NOMOGRAPH FOR FISH INGESTION EXPOSURE

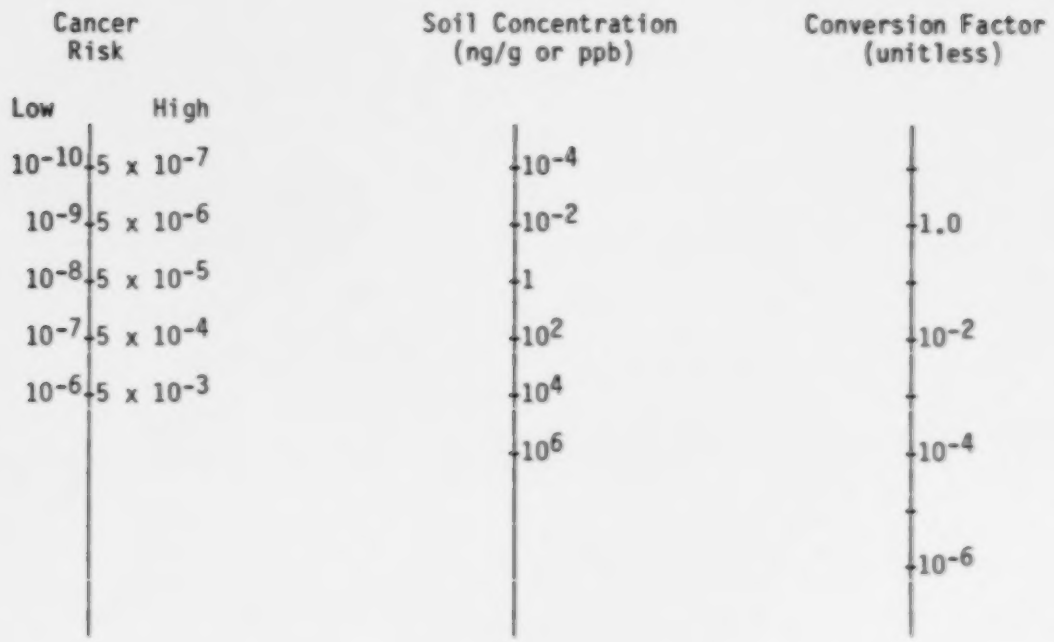


Cancer Risk = (Potency Factor x Soil Concentration x Conversion Factor x Exposure Duration x Fish Ingestion Rate x Fish-Sediment Distribution Factor x Absorption Fraction x Degradation Effects Ratio)/(Body Weight x Lifetime)

where, Potency Factor = .26-.31
Exposure Duration = 25,550 days
Fish Ingestion Rate = 6.5 g/day
Fish-Sediment Distribution Factor = 1-10 g soil/g fish
Absorption Fraction = .5-.86
Degradation Effects Ratio = .2-1
Body Weight = 70 kg
Lifetime = 70 yr

$$\text{Conversion Factor} = \frac{\text{Concentration of TCDD in Sediment Where Fish Are Caught (ng/g)}}{\text{Concentration of TCDD in Soil at Original Source (ng/g)}}$$

FIGURE A-3. NOMOGRAPH FOR DERMAL EXPOSURE

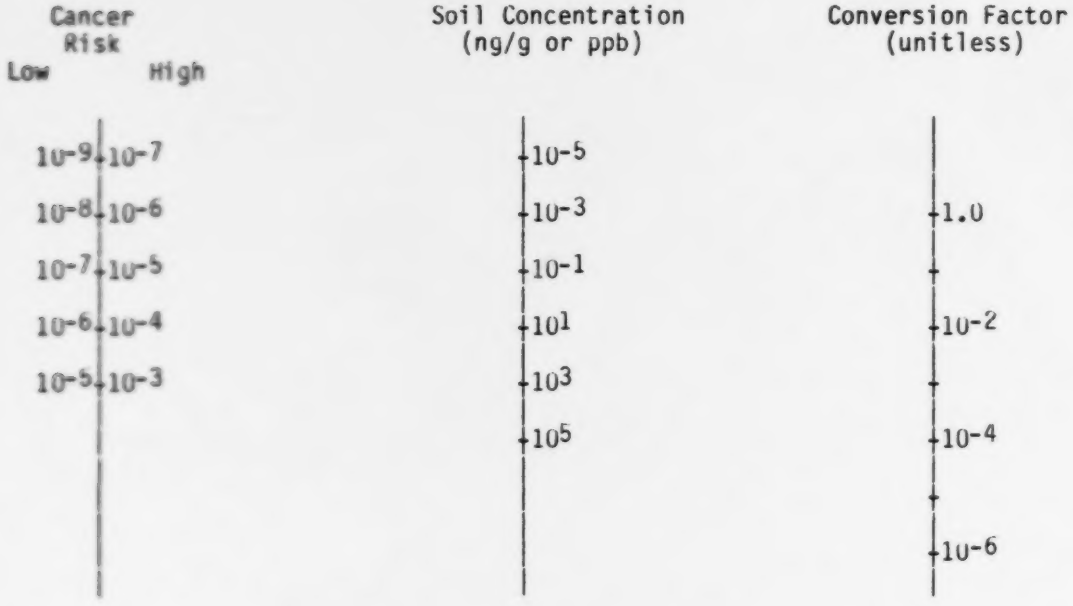


Cancer Risk = (Potency Factor x Soil Concentration x Conversion Factor x Lifetime Soil Accumulation x Absorption Fraction x Degradation Effects Ratio)/(Body Weight x Lifetime)

where, Potency Factor = .26-.31 $(ng/kg\text{-}day)^{-1}$
Lifetime Soil Accumulation = 7900-110,000 g
Absorption Fraction = .0007-.03
Degradation Effects Ratio = .2-1
Body Weight = 70 kg
Lifetime = 70 yr

$$\text{Conversion Factor} = \frac{\text{TCDD Concentration in Soil at Exposure Point (ng/kg)}}{\text{TCDD Concentration in Soil at Original Source (ng/kg)}}$$

FIGURE A-4. NOMOGRAPH FOR SOIL INGESTION EXPOSURE

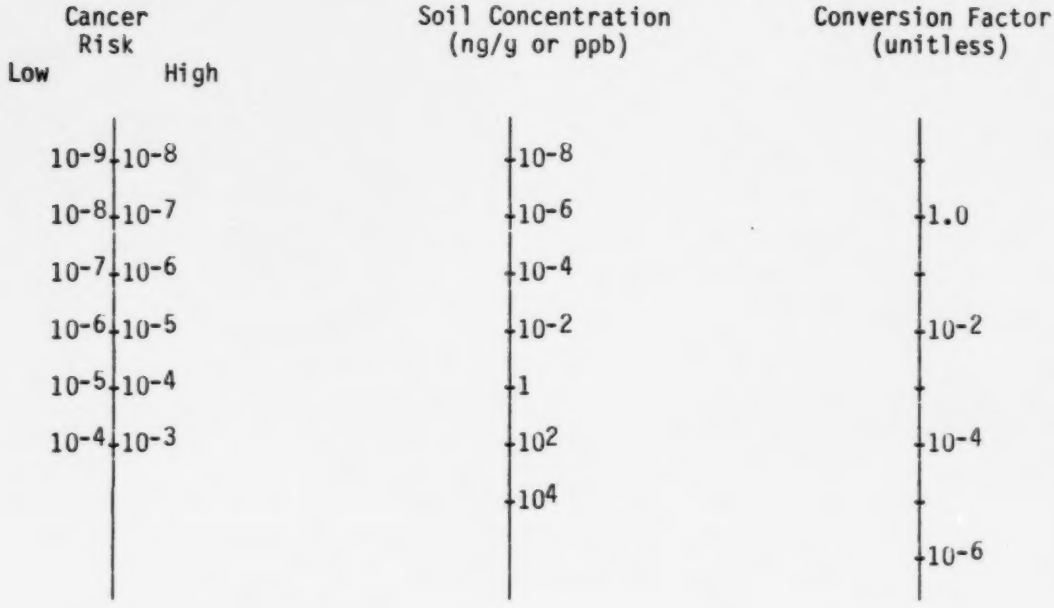


Cancer Risk = (Potency Factor x Soil Concentration x Exposure Duration x Conversion Factor x Soil Ingestion Rate x Absorption Fraction x Degradation Effects Ratio)/(Body Weight x Lifetime)

where, Potency Factor = .26-.31
Exposure Duration = 1240-1830 days
Soil Ingestion Rate = .1-5 g/day
Absorption Fraction = .2-.26
Degradation Effects Ratio = .84-1
Body Weight = 14 kg
Lifetime = 70 yr

$$\text{Conversion Factor} = \frac{\text{TCDD Concentration in Soil at Exposure Site (ng/g)}}{\text{TCDD Concentration in Soil at Original Source (ng/g)}}$$

FIGURE A-5. NOMOGRAPH FOR BEEF/DAIRY PRODUCTS INGESTION EXPOSURE



Cancer Risk = (Potency Factor x Soil Concentration x Conversion Factor x Exposure Duration x Beef/Dairy Products Fat Ingestion Rate x Animal Fat to Soil Bioconcentration Factor x Absorption Fraction x Degradation Effects Ratio)/(Body Weight x Lifetime)

where, Potency Factor = .26-.31 $(ng/kg\text{-}day)^{-1}$
Exposure Duration = 25,550 days
Beef/Dairy Products Fat Ingestion Rate = 62 g/day
Animal Fat to Soil Bioconcentration Factor = .4
Absorption Fraction = .5-.86
Degradation Effects Ratio = .2-1
Body Weight = 70 kg
Lifetime = 70 yr

$$\text{Conversion Factor} = \frac{\text{TCDD Concentration in Soil at Pasture (ng/g)}}{\text{TCDD Concentration in Soil at Original Source (ng/g)}}$$